Multiple Cystic Brain Infection: A Diagnostic Dilemma of Neurocysticercosis and Intracranial Tuberculoma - Case report

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Case Report

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Abstract

Background: Neurocysticercosis (NCC) is a central nervous system infection caused by Taenia solium, or pork tapeworm, affecting millions worldwide and possibly represents a leading cause of epilepsy in developing countries. NCC may be challenging to distinguish from intracranial tuberculomas, with tuberculosis highly prevalent in developing countries. We highlight the importance of clinical history, including exposure history and neuroimaging, in obtaining an accurate diagnosis to enable prompt treatment.

Case presentation: This case illustrates a 26-year-old gentleman diagnosed with NCC presenting with acute giddiness and headache. Clinical history suggested ingestion of undercooked pork, absence of constitutional symptoms, and neuroimaging demonstrated cerebral lesions with scolex and multiple lesions less than 2cm in diameter on brain MRI. He recovered well following a combination of oral albendazole and praziquantel, with corticosteroids prescribed as adjunct.

Conclusion:

This case highlights the salient features to distinguish NCC from intracranial tuberculoma. Early, accurate diagnosis will enable patients to obtain correct treatment and expedite recovery.

Background

Neurocysticercosis (NCC) refers to an infection of the central nervous system caused by cysticerci or larvae of the parasite Taenia Solium(1). Cysticercosis has been classified as a neglected tropical illness by the World Health Organisation (WHO) and affects an estimated 2.56 to 8.30 million individuals. Additionally, research indicates that approximately one-third of people with epilepsy residing in endemic regions are diagnosed with NCC(2). Bizhani et al.'s systematic review and meta-analysis found that prevalence in Southeast Asia ranges from 0.80% in Indonesia to 41.8% in Thailand, and globalisation has boosted prevalence in formerly low-prevalence areas(3). The overall incidence of NCC in Malaysia appears low due to underdiagnosis(4). This case study raises awareness concerning neurocysticercosis, enabling patients’ access to prompt and suitable therapy to mitigate its adverse consequences.

Case Presentation

A 26-year-old gentleman presented with giddiness for four days. It was acute in onset, first noticed while he was driving. It was associated with a continuous mild generalized headache with no postural or diurnal variation. He did not experience any fever, weight loss, or night sweats. He had a history of miliary tuberculosis and pneumothorax in 2017, which was treated with anti tuberculous drugs. He was clinically oriented on examination, pupils equal and reactive, afebrile, and vital signs were within normal limits. A complete neurological examination indicated no motor or sensory deficit, cerebellar signs or cranial nerve defects.
Complete blood count, renal and liver profile were unremarkable. Inflammatory markers were not elevated. Chest radiograph demonstrated reticulonodular opacities over both lung fields. Computed tomography brain imaging demonstrated scattered dense nodules with perilesional oedema surrounding two nodules at the left vertex. From lumbar puncture, opening pressure was 30 cmH2O, cerebrospinal fluid (CSF) protein was 312mg/L and acellular. Viral screening, toxoplasma IgM and Treponema Pallidum Hemagglutination were negative. Magnetic resonance imaging (MRI) of the brain demonstrated multiple bilateral cerebral and brainstem rim-enhancing lesions, the presence of scolex within the cyst and perilesional oedema over the left vertex.

Further history revealed he may have consumed undercooked pork. A diagnosis of NCC was made, and he was initiated on oral albendazole and praziquantel for two weeks. In addition, he was prescribed T. dexamethasone 7mg OD (0.1mg/kg/day) for two weeks, then tapered off gradually over a week. He was well after completing treatment with no complications.

Figure 1: Plain CT brain; axial cut showing calcified scolex within cyst (thick arrow) giving a 'target appearance'.

Figure 2: MRI brain; T2W coronal section showing left frontal cyst with surrounding perilesional edema (thin arrow).

Discussion

The life cycle of taenia solium, or pork tapeworm, is complex. It circulates in humans, the definitive host, and pigs, the intermediate host. The infection occurs following ingestion of cysticerci found in contaminated undercooked pork. The cysticerci develop into adult tapeworms and attach within the small intestine. Once matured, they produce proglottids and eggs, which pass out in faeces. Faecal-oral transmission of these eggs in areas with poor sanitation or polluted water can lead to cysticerci in multiple organs and NCC in the central nervous system(2). Unlike Taenia Solium taeniasis, Taenia Saginata taeniasis does not cause cysticercosis. Taenia Saginata taeniasis tends to be more symptomatic due to its larger size (5–25 meters) than Taenia Solium (2–7 meters long). It mainly manifests as gastrointestinal symptoms and the passing of proglottids. Rarely, migration of these proglottids may lead to appendicitis or cholangitis(5).

NCC typically presents as seizures or signs of elevated intracranial pressure. Clinical manifestations vary based on the location of the cysticerci. Typically, seizures, headache and giddiness are associated with parenchymal NCC. Similarly, giddiness was the primary presenting complaint in this instance. On the other hand, the most prevalent presentation of ventricular NCC is obstructive hydrocephalus, while subarachnoid NCC can present as meningitis, communicating hydrocephalus, stroke, or focal neurologic features. Mixed forms of NCC have also been frequently reported (8).

Detailed history and physical examination are crucial, emphasizing the exposure history, which should include ingestion of undercooked pork, sanitation in surrounding areas, access to clean water, contact
with tapeworm carriers, and pig stays (6). One critical differential to consider would be tuberculosis, which can mimic NCC. Symptoms such as fever, loss of weight, loss of appetite, or night sweats, which were not present in this case, must be enquired. Cervical lymphadenopathy should be examined as, if present, could suggest tuberculosis or mitotic lesions (7).

Once NCC is suspected, neuroimaging should be performed. NCC usually exhibits multiple calcifications, which can be easily detected through a Computed Tomography (CT) scan. Magnetic resonance imaging (MRI) can help differentiate different stages of NCC, detect scolex and perilesional oedema (6, 8, 9). In this case, the initial plain CT brain (Fig. 1) revealed scattered dense nodules with perilesional oedema surrounding two nodules at the left vertex. With the previous history of miliary tuberculosis, intracranial tuberculoma could not be ruled out until the scolex was demonstrated in subsequent MRI brain (Fig. 2).

Intracranial tuberculoma may resemble NCC in radio imaging, as they both present as rim-enhancing lesions (10). In this case, a lesion size of less than 2cm and the absence of signs of meningitis are indicators that intracranial tuberculosis is unlikely. Additionally, NCC affects the grey white matter, whereas intracranial tuberculoma commonly affects the brain's posterior fossa. On MRI T2W, NCC appears hyperintense with a hypointense scolex and no midline shift, whereas tuberculoma is hypointense and accompanied by a midline shift (10).

Our patient was treated with oral albendazole and praziquantel for two weeks. A recent randomized trial showed that combination therapy was superior to albendazole monotherapy in radiologic resolution of the lesion. A fundoscopy examination is vital to rule out intraocular cysticerci, as antiparasitic agents may result in blindness following inflammation due to the degradation of cysts. The concomitant use of anti-inflammatory agents or corticosteroids reduces such risks and hastens recovery.

**Conclusion**

Neurocysticercosis and intracranial tuberculoma can be challenging to differentiate. In this case, we demonstrated that a thorough clinical history emphasizing exposure history, physical examination and neuroimaging modalities aid in clinching the diagnosis. Ingestion of undercooked pork, absence of constitutional symptoms and meningism, presence of scolex, and the size of lesions < 2cm in brain MRI were the key characteristics in differentiating both diseases.

**Abbreviations**

NCC  
Neurocysticercosis

**Declarations**

- Ethics approval and consent to participate - not applicable
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References


Figures
Figure 1

Plain CT brain; axial cut showing calcified scolex within cyst (thick arrow) giving a 'target appearance'.
Figure 2

MRI brain; T2W coronal section showing left frontal cyst with surrounding perilesional edema (thin arrow).